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CONTENTS

ANNUAL GRADUATE FORTNIGHT: "DISORDERS OF THE CIRCULATION," October 19 to 30, 1931.. 47

Arteriosclerosis 47

A List of Writings on the Cardiovascular System Exhibited during the Fortnight..... 66

Recent Accessions to the Library..... 94

Proceedings of Academy Meetings..... 98

Fellows Elected February 4, 1932..... 104

Deaths of Fellows of the Academy..... 105

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THE NEW YORK
ACADEMY OF MEDICINE

VOL. VIII FEBRUARY, 1932 No. 2

ANNUAL GRADUATE FORTNIGHT
Disorders of the Circulation
October 19 to 30, 1931

ARTERIOSCLEROSIS*

Roy Wesley Scott
Professor of Clinical Medicine, Western Reserve Medical School

I undertake this discussion of human arteriosclerosis with a certain unwillingness, because as every student of the subject must appreciate, our ignorance of this very common affection far overshadows our knowledge. A search of past writings for established fact leads to disappointment; hypotheses there are in abundance, but these frequently express opposite views concerning the cause and nature of arteriosclerosis.

For the purpose of this discussion I exclude those changes in the arteries due to syphilis, tuberculosis, trauma, toxic agents leading to thrombosis, and that rather rare inflammatory process, periarteritis nodosa. With less justification perhaps we exclude also the medial necrosis and calcification of peripheral vessels, described by Mönckeberg. I shall use the older and better known term "arteriosclerosis" introduced by Lobstein to denote that most common affection of the arterial system, of undetermined etiology, rarely absent in advanced life, and characterized by a thickening of the intima accompanied by the deposition of fat. Changes in the media are usually mild, although in some instances they are marked. Later sclerosis and calcification occur, which give to the arteriosclerotic artery its most characteristic appearance.

*Delivered October 22, 1931.

To enter upon a discussion here of the anatomical changes in arteriosclerosis would carry us far afield and would be productive of nothing new. Whether the process is primarily in the intima or in the media, or whether it is inflammatory or degenerative in nature is not settled. Klotz thinks that the sclerotic changes in the vessels are due for the most part to intimal disease caused by infectious processes, work and old age, while McCallum has stated that the media in the aorta and visceral arteries seems relatively little changed in comparison to the extent of the intimal alterations. Included here are those changes in the smaller ramifications of the vascular tree known as arteriolar sclerosis. No attempt is made to differentiate this process from arterial sclerosis because the two are very frequently associated and so far as we now know they may have a common pathogenesis.

Arteriolar sclerosis affecting arterioles of the order of the vasa afferentia in the kidneys is characterized by the irregular deposition of hyalin under the endothelium as seen best in longitudinal sections of the arteriole. Such hyalinized areas may undergo a fatty change which can be demonstrated by lipoid stains such as Sudan III. As the process advances the lumen of the vessel is narrowed or completely obliterated. In larger arterioles one sees only the hyperplastic type of intimal thickening with retrogressive and fatty changes and later proliferation of connective tissue. Often in advanced cases the media shows degenerative changes with complete replacement fibrosis. Medial hypertrophy of the kidney arterioles is seen particularly in individuals under forty dying of hypertension.

Reviewing the present day conceptions of the etiology and pathogenesis of arteriosclerosis, one finds much speculation and many hypotheses often masking as fact. In a review of the subject in 1922, McCallum stated: "Arteriosclerosis is one of those diseases difficult to explain because it develops so slowly through long years of life, during which a great many possible causes have had an opportunity to affect the tissues. Hence every conceivable idea

has been expressed or tenaciously maintained regarding the condition and many of them are so vague and ill supported that it is wearisome to discuss them. None, however, is clearly demonstrated to have a definite bearing on the etiology of arteriosclerosis and we are quite as ignorant of its underlying causes as were our fathers in the days of Morgagni."

Aware of the truth expressed in this quotation of McCallum, we find it difficult however to dismiss summarily from consideration certain factors which have long been thought to be of some significance in the cause of human arteriosclerosis. I refer to the influences of heredity, to the ravages of the ageing process, to hypertension (high blood pressure) and to those changes in our environment incident to our civilization, not because these are in any sense all the factors that may be involved, but the evidence in their favor is perhaps open to less criticism than that presented by other alleged etiological agents.

Incidence of Arteriosclerosis

Accurate data on the present incidence of arteriosclerosis are not available, but indirectly, certain more or less pertinent facts are obtained by a study of mortality and morbidity statistics. These show for the registration area in America, first, that heart disease is the leading cause of death at present and, second, that the mounting incidence of heart disease during the past two decades occurs in individuals past forty years of age. For example, heart disease in the State of New York, excluding New York City, has been the leading cause of death since 1912 with the exception of the year 1918 when pneumonia held first place. The curves show an almost continuous rise from a rate of 133.5 in 1900 to 279.9 in 1928, when 50.6 per cent of the deaths were between the ages of forty and seventy, and only 8.3 per cent were under forty years of age. Assuming a certain error inherent in such data, there is little reason to doubt the statement that the most significant factors in heart disease beyond forty are vascular (blood vessel) decay, coronary arteriosclerosis, and those changes in the

smaller arterioles that we know are so frequently associated with hypertension (high blood pressure). If, in addition, we add that group of individuals beyond forty who die of cerebral arteriosclerosis (thrombosis or hemorrhage) and those dying of renal insufficiency (kidney disease) secondary to vascular disease, it is apparent that at the present time the expectancy of life of the majority of people beyond middle age is determined by the state of the arteries. Long ago it was said "A man is as old as his arteries." This truth is even more apparent today than ever before.

Much attention is given in some quarters to the steadily mounting morbidity and mortality rates from cardiovascular (heart and blood vessel) disease, and a variety of opinions is expressed concerning their significance; but before any definite conclusions are reached it is necessary to know much more than we do at present concerning many factors that appear to influence the incidence of arteriosclerotic changes in vessels.

Data on the life span show that there are today in this country more people fifty years of age than ever before. Only a few decades ago the age at which most people died was 45; now it is 60. The control of some of the important causes of death in infancy and childhood has increased the number of people past middle life—in the arteriosclerotic age—hence a certain natural increment in the incidence of vascular disease is to be anticipated. Furthermore, modern hygiene and preventive medicine are striving daily to increase the number of individuals (both fit and unfit) that mature to approach the involutionary period of senescence. Man is exercising more and more control over his environment and there are those who point with pride to his accomplishments in prolonging human life, but the Biologist and the Eugenist ponder the ultimate results of our present day methods as applied to the human race. They see the waning influence of natural selection and wonder if we can continue to thwart nature without paying the penalty in quality if not ultimately in quantity. In fact, there is some reason to believe that

nature is already exacting her toll. Professor Forsyth of Dartmouth has presented evidence to show that the length of life in this country is actually decreasing. From a critical analysis of mortality statistics he concludes that the American adult today cannot expect to live as long as his father or grandfather. There is little question that many individuals today are maturing to propagate the race who would not have survived in the more rigorous environment of our forefathers. If the wearing quality of our arteries is in any way determined by our heredity (a point emphasized by the late Dr. Osler) may we not expect an increasing number of people whose vessels may wear reasonably well for the earlier decades of life but as age comes on the shoddy appears in the form of early deterioration of the vascular system. It appears that this view, admittedly difficult either to prove or disprove, cannot in the light of our present knowledge be summarily dismissed as sheer speculation.

Clinical Aspects of Arteriosclerosis

Although we are far from a solution of the problem of the pathogenesis of human arteriosclerosis, valuable contributions to our knowledge have been made by both pathologists and clinicians in the days since Bright. Time will not permit a discussion of the voluminous literature that has grown up on the subject in the past century, but it is necessary to mention the contributions of a few workers who have been most responsible for our present day conceptions. These are not perfect and they are slowly changing, but there is little doubt that the informed clinician at present is much better equipped than were his predecessors of the past generation to appreciate the variety of clinical conditions either associated with or directly due to arteriosclerotic changes in the vascular tree.

Knowing as we now do the frequent association of hypertension and left ventricular hypertrophy with sclerotic changes in the intimate vasculature of several organs—most often the kidneys—it is not surprising that the early students of kidney disease should have been

concerned also with the problem of vascular disease. Bright observed the occurrence of thickened vessels and cardiac hypertrophy in patients with contracted kidneys, but he had no clear conception of the causal relation. To understand the origin of the term "Bright's Disease" and "Bright's Kidneys" and to appreciate the influence that these terms have had on moulding our views even to the present time, we should recall that Bright's work dealt with no particular form of renal disease but included all cases with an albuminous urine during life and a scarred kidney post mortem. Kidney lesions were no further classified by Bright than as "large and smooth" and "small and granular," exceptions being regarded as intermediate stages in the process of development from the large to the small kidney. Originally, therefore, the term Bright's Kidney included many forms of renal disease. It is interesting to note that following Bright's work and persisting for many years were certain conceptions regarding kidney disease which have done more to retard than to advance our knowledge. Chief of these were: First, that all types of scarred kidneys were manifestations of one disease process at different stages, and, second, that the associated lesions of other organs, such as those of the heart and arteries, were the effects of renal disease and due to progressive renal insufficiency.

It was not until some twenty years after Bright that the vascular lesions associated with renal disease received much attention. In 1852 Johnston showed that in one form of chronic Bright's Disease the smaller arteries not alone in the kidneys but also in other viscera were thickened—changes which he ascribed to antecedent renal disease. The contribution which marked an epoch in our knowledge of vascular disease was that of Gull and Sutton in 1872. They confirmed Johnston's observations, but took an important step forward by showing that a diffuse vascular disease of the smaller vessels (arteriocapillary fibrosis) may occur without serious renal damage. They further suggested that left ventricular hypertrophy was due

to the change in the minute arteries and capillaries and not to renal insufficiency. The observations and deductions of Gull and Sutton are all the more remarkable when we recall that they were made with imperfect histological methods and with no knowledge of clinical blood pressure. As conclusive as their work appeared, it was not a death blow to the older conceptions of the dependence of cardiac hypertrophy and arteriosclerosis upon renal disease.

About twenty years after the work of Gull and Sutton, the sphygmomanometer was introduced into clinical medicine by Von Basch. As clinical data on blood pressure gradually accumulated it was but natural that some association between abnormal elevations in blood pressure and sclerotic changes in vessels should be postulated, and supporting evidence for this view was afforded by those incidences of hypertension associated with demonstrable sclerosis of the accessible arteries. However, exceptions to the rule were not long in appearing. Von Basch in 1893, after observing many cases of hypertension without demonstrable arterial changes, concluded that the hypertension was but a precursor of arteriosclerosis and to designate such conditions he used the term "latent arteriosclerosis." Still lurking in the background now more than twenty years after Gull and Sutton's work, and more or less dominating the clinical thought of the time was the old teaching concerning the unity of arteriosclerosis and kidney disease. To bring the earlier observations on blood pressure into line with this view, it was taught that hypertension was a manifestation of arterial change which in turn resulted from renal disease. Therefore individuals with an abnormal elevation in blood pressure were regarded as having, or destined to develop Bright's Disease and renal insufficiency. Such was the generally accepted and almost universally taught hypothesis concerning the relationship between arteriosclerosis, blood pressure, and kidney disease, in the early 90's of the last century.

In retrospect we may say that the stage was set for recognition of that large group of individuals with chronic

hypertension who never develop renal insufficiency, and who have no significant changes in the palpable arteries. For this step forward we are indebted to Allbutt, although about the same time Huchard in France was aware of the frequency of non-nephritic hypertension. Long and careful observations on patients led Allbutt to the following well known classification: (1) Hyperpiesia—individuals with chronic hypertension with little or no renal involvement who die of heart failure or apoplexy. (2) Renal disease with or without hypertension. (3) Decrescent arteriosclerosis, primarily of the larger arteries, not necessarily accompanied by hypertension. In his zeal to stress the importance of hyperpiesia and to have it regarded as a distinct malady, Allbutt failed to recognize, or at all events did not include in his classification, those cases of primary hypertension that die of renal insufficiency. Such cases have until very recently presented a barrier to any classification of nephritis; they have led to no little confusion between clinicians and pathologists and have helped to keep alive the old conception of the renal origin of hypertension.

After Gull and Sutton, the most outstanding contribution from the pathological standpoint was made by Jores in 1904, who showed for the first time that the scarred kidney, formerly regarded as the seat of chronic interstitial nephritis, was in reality due to arteriosclerosis of the smaller renal vessels. He further observed that patients with atrophic granular kidneys had hypertrophied left ventricles, and more often died of cardiac failure or apoplexy. Jores' work suppor ed that of Allbutt and served to emphasize the view tha t cardiac hypertrophy and hypertension were not due to renal involvement. Opponents on the other hand supported their argument by citing cases of hypertension that died of uremia. For several years following the work of Allbutt and Jores clinicians generally, recognized that some relationship existed between arteriosclerosis, hypertension, cardiac hypertrophy and granular kidneys, but, as most of us recall, the teach-

ing of twenty years ago was anything but clarifying. The diagnosis of cardiovascular renal disease was commonly made, but so far as the student was concerned, just what part of the clinical picture was due to cardiac failure and what to renal insufficiency was seldom determined with any degree of accuracy. Retained body fluid and an albuminous urine were usually sufficient evidence to incriminate the kidneys.

The clear recognition of two types of primary hypertension, one with renal insufficiency, the other without, and the corresponding vascular renal lesions were established by Volhard and Fahr in 1914. Thus for the first time the hitherto puzzling case of hypertension and renal insufficiency was brought into line, but not without an argument among pathologists which has continued to the present time. This arose from the assumption by Volhard and Fahr that the kidney lesions, particularly in the glomeruli, in their cases of so-called malignant hypertension dying of renal insufficiency, were not due solely to vascular damage but were a combination of vessel and inflammatory renal changes—their Kombinations-Form. Both authors have now changed their original views, Volhard holding that the vascular renal lesions are ischemic in nature, the result of vascular spasm. This phase of the subject is presented in a recent contribution of Klemperer and Otani, who distinguished two types of renal insufficiency in hypertensive patients, first, a slowly progressive one due to gradual stricture of the vascular bed with subsequent destruction of kidney parenchyma, and, second, the so-called malignant hypertension with rapidly developing renal insufficiency in which marked renal atrophy is absent. In this latter group Klemperer and Otani distinguish two types of vascular lesions, first, a rapidly progressive atherosclerosis and, second, an inflammatory lesion superimposed on an antecedent arteriosclerosis.

The above brief resume of a few of the more important contributions since Bright, gives us a glimpse of the development of our present knowledge concerning the rela-

tionship of arteriosclerosis, hypertension and certain forms of kidney disease, and it also indicates the difficulties encountered in the past in correlating clinical and pathological observations. That our present knowledge is immature is apparent to all, but may not its abundant heritage promise much for the future?

Before attempting what we may call a pathological-physiological classification of arteriosclerosis, certain well known facts may be emphasized: First, that those changes in the arterial tree that we call arteriosclerosis are most capricious in their distribution, appearing in the greater circulation from the Sinus of Valsalva to the smallest arterioles (for some reason not known at present the arteries of the lesser circulation in most cases are singularly spared); the process may be generalized in some instances and sharply localized in others. Second, that the functional significance of arteriosclerosis varies greatly —often there is no parallelism between the extent of the process and the associated functional disturbances. For instance, we see extensive changes in the larger arteries that offer no barrier to longevity, and on the other hand, the process may be confined to a few millimeters of the coronary artery and cause sudden death in the prime of life. Serious damage to the intimate vasculature of the kidney is found in young people dying of uremia. Chronic hypertension and sanguineous apoplexy usually are associated with damaged cerebral vessels. Many other examples might be cited to illustrate the variety of clinical pictures associated more or less intimately with vascular disease. Our limited knowledge at present precludes a satisfactory classification of arteriosclerosis (this term to include both large and small vessel involvement), but since we as clinicians are primarily concerned in correlating the clinical and pathological findings, the following scheme may serve as an outline in approaching a discussion of the subject:

PATHOLOGICAL PHYSIOLOGY OF ARTERIOSCLEROSIS

Arteriosclerosis (large arteries)

Without Hypertension

(1) Coronary Arteries
- Stenosis (ischemia) → Angina Pectoris
- Thrombosis → Myocardial Insufficiency

(2) Cerebral Arteries
- Stenosis (ischemia) → Mental Deterioration
- Thrombosis → Apoplexy / Paralysis
- Rupture → Apoplexy / Paralysis

(3) Renal Arteries
- Ischemia / Necrosis / Infarction → Scarred Kidney → very rarely insufficient

(4) Aorta
- Inelasticity with or without dilatation → Peripheral Circulatory Disturbances
- Calcification of Aortic Valves → (Stenosis) Myocardial Insufficiency

Arteriolosclerosis (arterioles)

Often combined with large vessel sclerosis
With Hypertension and Cardiac Hypertrophy

(5) Heart—
- Dilatation → Myocardial Insufficiency
- Coronary Disease as in (1) → Angina Pectoris

(6) Brain—
- Vascular Changes as in (2) → Mental Deterioration
- Apoplexy / Paralysis

(7) Kidney—
- Vessel Sclerosis with or without Arteriolar Necrosis → Often Granular Atrophy → Uremia in approximately 10%

This outline makes no claim to perfection and it has many vulnerable spots. To perceive them is to appreciate the difficulties in associating functional disorders and anatomical changes, particularly changes so irregular in distribution as are those of arteriosclerosis. Certain quite obvious objections may be made to the association of arterial and arteriolar sclerosis and blood pressure, but in indicating a relationship between hypertension for example and small vessel disease, no more is implied than the fact, that most individuals with so-called essential hypertension show at post mortem more or less marked lesions of the arterioles. We have no proof that the arteriolar lesions are the cause or the result of hypertension and no such implication is intended in this outline. As a matter of fact there are authorities who uphold either view.

Lesions of the arterioles are found in the order of frequency and severity in the kidney, spleen, pancreas, liver, brain and gastro-intestinal tract. Keith and associates at the Mayo Clinic recently have published observations on the arteriolar lesions in the skeletal muscles in cases of hypertension. This work seems promising but more data are necessary before definite conclusions can be drawn. The occurrence of large vessel sclerosis without hypertension as shown here is also open to criticism because of the frequent involvement of the arteries as well as the arterioles in individuals with hypertension. On the other hand, one finds it necessary to include that large group of elderly people who have outspoken arteriosclerosis of the large arteries with no undue elevation in blood pressure. Certain it is that there is not the obligate connection between large vessel sclerosis and hypertension that we know exists between small vessel disease and hypertension. For this reason there appears some justification for indicating the relationship as shown here. The right side of this diagram showing items 5, 6, and 7 under "Small Arteries" and in relation to hypertension is a functional grouping emphasizing a fact well established at present, namely,

that the cause of death in individuals with primary or essential hypertension is in the order of frequency: heart failure, a cerebral accident and renal insufficiency. In a series of 400 autopsied cases of essential hypertension, Bell and Clawson found myocardial insufficiency including coronary disease, responsible for sixty per cent of the deaths. In a similar series of 100 autopsied cases at the Cleveland City Hospital we found as the cause of death myocardial failure in sixty-eight per cent, cerebral accident in twenty-two per cent, and renal insufficiency in ten per cent. Allowing for certain errors inherent in such data, they show that the most common contingency in essential hypertension is heart failure, and by all odds the least is renal insufficiency. These facts are not as widely appreciated as their significance merits. The old conception of the renal origin of hypertension still survives to influence diagnosis and the therapeutic management of patients. Red meat is still prohibited and a failing heart is neglected because an albuminous urine is regarded as Bright's Disease rather than as chronic passive congestion. The appearance of coronary arteriosclerosis in two positions in this diagram illustrates again the difficulty in correlating anatomical changes and functional disorders. Anatomically the coronaries are large arteries and from a functional standpoint coronary sclerosis occurs in patients without hypertension, but it also is associated frequently with small vessel disease and hypertension. Indeed, more or less coronary arteriosclerosis is almost always found in hypertensive heart failure, so that, in a scheme such as this, it must appear on the one hand associated with hypertension, and on the other hand without hypertension. For the same reason, we must include cerebral arteriosclerosis under the two headings. The arrangement in 5 under "Small Arteries" is made to indicate, first, that coronary arteriosclerosis frequently occurs and is often an important factor in hypertensive heart failure, and, second, that as a result of coronary disease the clinical picture in hypertension may be that of Heberden's Angina Pectoris or coronary thrombosis. In 6 we see the second

most common cause of death in hypertension—a cerebral accident due to a ruptured cerebral artery or to thrombosis of a sclerotic vessel. Under 7 we include that group (approximately ten per cent) of the cases of hypertension that die of renal insufficiency. Here we may distinguish two general types, one of long standing hypertension—a slowly progressive vascular lesion in the kidney with renal atrophy and ultimate insufficiency—the other a more rapidly developing vascular lesion, usually in younger individuals, with little or no renal atrophy, and death in uremia. In group 7 are included those instances of granular atrophy of the kidney secondary to vascular lesions, so commonly found at autopsy in hypertensive patients. Such forms of renal scarring which up to a few years ago were called chronic interstitial nephritis we now know, seldom lead to renal insufficiency. In other words, from eighty-five to ninety per cent of patients with essential hypertension and arteriolar sclerosis die of heart failure or a cerebral accident before the vascular lesion in the kidney progresses to the point of renal insufficiency. The left half of this outline is made to include those instances, usually in elderly people, of widespread and often extensive changes in the larger arteries without hypertension. Here reference is made to four large arterial systems, because the functional disturbances associated with changes in these vessels often lead to fairly distinct clinical pictures. For example, coronary arteriosclerosis is seen as the anatomical basis for certain cases of myocardial fibrosis and functional insufficiency, for instance of angina pectoris and coronary thrombosis in patients who have never had hypertension. Also the mental deterioration in encephalomalacia from cerebral arteriosclerosis is well known, as is also the apoplectic stroke and paralysis.

The kidney is so often scarred in arteriosclerosis of the larger arteries that we include it under (3). Changes in the larger vessels of the kidney often lead to occlusions with atrophy of kidney substance. This causes the irregular pitting so often seen in the senile kidney. Such

changes, however, seldom cause atrophy of sufficient kidney parenchyma to lead to functional insufficiency.

Sclerosis of the aorta may be extreme and yet cause no symptoms. However, as the vessel approximates rigidity the pulse is more rapidly propagated to the periphery, as shown by Bramwell and Hill. More of the shock of ventricular systole is transmitted peripherally and we find an abnormal excursion of the vessels from the aortic arch, for example the carotids. Sclerotic changes at the root of the aorta not infrequently involve the valve cusps, which become rigid, are unable to approximate the aortic wall during systole, and may lead to a high grade of aortic stenosis and death from heart failure. Incidences of aortic insufficiency ascribed to arteriosclerosis are often due to syphilis which may not be recognized grossly in the presence of advanced arteriosclerosis. Mention here may be made of an interesting anatomical finding first called to my attention by Professor Erdheim. He points out that the first few centimeters of the aorta are singularly spared in individuals who attain seventy years or more, although the remainder of the vessel is more or less extensively diseased. On the other hand, marked involvement of the first 3 to 4 centimeters appears to be incompatible with the proverbial three score and ten years. Exceptions to this rule are very rare. In searching for some explanation of this fact, one is reminded of the evidence indicating that the first part of the aorta is nourished from the coronary arteries, so that it is possible some relationship between coronary disease, at least of those branches that supply the aortic root, and sclerosis in this region exists. This matter merits further study.

The reference to combined lesions, as indicated in the right half of this diagram, is of course absolutely necessary, since several combinations of functional and anatomical changes may occur. For example, the larger arteries are almost always involved in long standing cases of hypertension and, indeed, the clinical course may be dominated entirely by the large vessel disease.

At this point I desire to present briefly the salient clinical and pathological findings in one case which illustrates the extent of the problem of vascular disease. Those of you who attended the Cleveland meeting of the American College of Physicians in 1927, will recall a boy aged twelve years whom I demonstrated as an example of juvenile non-renal hypertension.

This boy developed normally and except for an attack of lobar pneumonia at age eight, he was in good health until eleven years of age. There was no history of rheumatic infection or scarlet fever. One year before coming under our observation he noticed undue breathlessness and palpitation while at play. He complained also during this year of rather regularly recurring attacks of severe headache. These symptoms progressed and formed the chief complaint on admission to the hospital, January 20th, 1927.

Physical examination revealed a well developed and alert boy of good color, in no distress, and with no puffiness about the face or edema elsewhere. Blindfolded, one would have said from palpation that the accessible arteries were those of an old man. They were hard and tortuous and it was apparent that the blood pressure was elevated, and when measured it was found to be 280 mm. of mercury systolic, and 175 diastolic. At a glance one was impressed by the vigor and position of the cardiac impulse, which was maximum in the sixth left intercostal space, extending almost to the anterior axillary line. At the apex the heart sounds were loud and booming, but as one approached the base a loud diastolic murmur was audible, with maximum intensity over the manubrium. In this position one also heard a snappy and tympanitic second sound followed at once by the blowing diastolic murmur. It was thought that this murmur was due to relative incompetence of the aortic valves, as it entirely disappeared when the systolic blood pressure was lowered from 280 to 190 by the administration of nitro-glycerine. It is

of note also that this acute drop in blood pressure always caused a severe headache.

The eyegrounds were typical of hypertensive retinitis. Neither disc was discernible from swelling of the adjacent retina. There were numerous areas of recent hemorrhage and a few whitish spots were observed. Only a few retinal arterioles were visible. These showed marked sclerosis. Further clinical study of the case revealed a normal blood picture, hemoglobin 90 per cent with 4.3 million red blood cells per cubic-millimeter. The blood urea nitrogen on several occasions varied between 40 and 60 mgm. per 100 c.c. The urine was pale and the specific gravity varied between 1.010 and 1.015. It always contained albumin and the sediment showed a few casts. The phthalein output was 50 per cent in two hours.

During the first month in the hospital, the symptoms complained of on admission improved, and the child appeared so well that his parents insisted upon taking him home. Two weeks later, and while at home, he developed suddenly a right sided hemiplegia with the signs of an upper neurone lesion of the seventh nerve. Two days afterwards he was readmitted to the hospital, but no improvement in his condition occurred. His paralysis continued, he became more and more lethargic, and expired on April the 5th, two and a half months after his first admission. The anatomical diagnosis was as follows: Marked Generalized Arteriosclerosis; Arterial and Arteriolar Sclerosis of Kidneys; Cardiac Hypertrophy (weight 475 grams); Massive Cerebral Hemorrhage, Left; Hemorrhagic Infarcts of Intestines; Acute Splenic Hyerplasia; Edema of Lungs. Microscopically, arteriolar lesions were demonstrated in the kidneys, liver, spleen and pancreas. For the following histological description of the kidney lesions I am indebted to my colleague Dr. Harry Goldblatt. One of the striking features is the relative absence of glomerular lesions. An occasional glomerulus shows fatty degeneration or focal hyalinization of the tuft, with or without adhesion to the capsule. The most striking pathological

change affects the blood vessels and especially the interlobular and afferent vessels. In the larger arterioles there is mainly thickening of the intima due to cellular proliferation. In the smallest ones the thickened, proliferated intima shows patchy or complete hyalinization with or without fatty degeneration; which shows well in sections stained with Sudan III. The media of the smaller vessels is definitely hypertrophic, but in most instances shows no other pathological change. The vascular lumen is in all instances reduced in size and in many of the vessels it is completely obliterated.

From the above account we see a case of extensive and severe diffuse vascular disease with hypertension and death from a cerebral hemorrhage, in a boy of twelve years. Certainly we cannot invoke the ageing process, and there is little evidence that infection played an etiological rôle in the production of the vessel disease in this case.

In summary form we may say that the present day clinician sees in human arteriosclerosis a pathological process affecting the arterial system of the greater circulation from the largest to the smallest vessels, usually insidious in its onset, and most irregular in its distribution. Its pathogenesis is an unsolved problem, but in speculating we turn to the influence of such factors as heredity, the ageing process, hypertension, and those changes in our environment incident to modern civilization. When we know more about the causes of essential hypertension we will be farther on the road to the solution of the problem of arteriosclerosis, particularly the form that involves the smallest vessels, the arterioles.

We have seen that statistics indicate a mounting incidence of vascular decay, particularly in middle life, but we are reminded of the fact that individuals are living longer today than ever before and deterioration of the arteries appears to be a favorite method of nature to eliminate us after our biological responsibilities have been fulfilled. Complicating the problem and exerting an influence diffi-

cult to evaluate, are our present day efforts to thwart the law of natural selection.

One is reminded of the proverb, "So long as more remains to be done, nothing seems done." However, the canvas is being painted, a high light here, a shadow there, and if we are at present forced to speculate on the appearance of the final picture, we should be stimulated rather than deterred in our efforts to solve one of the most important and most perplexing problems with which the clinician and pathologist have to deal—the problem of vascular disease.

A LIST OF WRITINGS ON THE CARDIOVASCULAR SYSTEM EXHIBITED DURING THE GRADUATE FORTNIGHT OF THE NEW YORK ACADEMY OF MEDICINE, 1931

Arranged by
Dr. Charles K. Friedberg
and the Staff of the Library.

In connection with the program of the Graduate Fortnight on the cardiovascular system and its disturbances, organized by The New York Academy of Medicine in October, 1931, an exhibit was arranged of books and articles epitomizing the historical development of this subject. The more valuable volumes and single articles were opened at significant illustrations or paragraphs, and placed in closed glass cases with an associated card of explanation. More general volumes such as reference books, textbooks on heart disease, and books on special branches of the subject, were exhibited on open book shelves where they were easily accessible to visiting physicians. The books and articles on the open and in the closed shelves were demonstrated and discussed by the exhibitor.

Because of limitations of space it was impossible to include all the important works contributing to progress in cardiovascular disease. Availability and special interest were often the determining factors in choice of material. Related subjects such as hypertension, thyroid heart, etc., were omitted because of restricted space and because it is planned to consider these subjects in greater detail at future graduate fortnights.

In the preparation of the exhibit we wish to acknowledge our thanks to Dr. Fielding H. Garrison and Dr. Emanuel Libman for numerous valuable suggestions. We wish to acknowledge our indebtedness also to Sir Humphry Rolleston's Harveian Oration (1928) on "Cardiovascular disease since Harvey's discovery," H. L. Flint's

"The heart: old and new views," (1921), and R. O. Moon's "Growth of our knowledge of heart disease," (1927), for bibliographic material. We want to thank Dr. Emanuel Libman and Dr. Eli Moschcowitz for the loan of several volumes.

C. K. FRIEDBERG.

EARLY HISTORY

SINGER, CHARLES JOSEPH (1876-).

The Evolution of Anatomy.

London, K. Paul . . ., 1925.

The illustrations opposite page 3 show paleolithic drawings of elephant and Magdalenian drawings of bison indicating position of the heart.

HIPPOCRATES (B. CA. 460 B. C.).

Oeuvres complètes . . . Traduction nouvelle . . . par E. Littré.

Paris, Baillière, 1839-1861. 10 v.

Vol. VI opened at p. 44-45 in section on the nature of man: when life was seen to ooze away in wounded people, the blood was naturally mistaken for the soul of man.

ARISTOTELES (CA. 384-322 B. C.).

De Animalibus . . . Translated [into Latin] by Theodorus Gaza.

Venice, Johannes de Colonia & Johannes Manthen [de] Gherretzem, 1476.

A beautiful copy of one of the finest volumes from the press of these noted printers. Opened at passages indicating that unlike Hippocrates who regarded the brain as the seat of sensation, Aristotle located this in the heart which he viewed as the "Acropolis of the body" (De Partibus Animalium, III).

GALEN (CA. 129-200 A. D.).

De Naturalibus Facultatibus. Vol. II of Kühn's Medicorum Graecorum Opera . . . Leipzig, Cnobloch, 1821.

Describes, p. 208-209, the ventricular septum which he believed contained minute pores through which the blood is attracted from the right to the left ventricle during diastole.

PAGEL, JULIUS LEOPOLD (1851-1912).

Einführung in die Geschichte der Medizin.

Berlin, Karger, 1915.

Diagram of the Galenic circulation, p. 121. Note particularly the direct communication of right and left ventricles by a permeable septum, and the vessels passing directly from the liver (c) to the rest of the body as well as back to the heart.

HIPPOCRATES (B. CA. 460 B. C.).

Oeuvres complètes . . . Traduction nouvelle . . . par E. Littré.

Paris, Baillière, 1839-1861. 10 v.

Vol. IX opened at p. 83 in section on the Heart: "The heart is a muscle."

DEVELOPMENT OF ANATOMICAL KNOWLEDGE.

DA VINCI, LEONARDO (1452-1519).
Quaderni d'Anatomia.
Christiania, Dybwad, 1911-1916. 6 v.
Famous drawings of cardiac structures. (Originals now in Windsor Castle). Contains accurate descriptions and drawings of cardiac valves (Vol. II opened at folio 8 verso). Belief in permeability of septum indicated by drawings exhibited in Vol. I, folio 3 recto. Da Vinci stated, however, that the pores were invisible. Drawings include illustration of the moderator band.

VESALIUS, ANDREAS (1514-1564).
De Humani Corporis Fabrica. First edition.
Basle, ex off. Oporini, 1543.
Inauguration of the renaissance of Anatomy. In Book VI, Chapter xi, p. 567 (see figure), he describes the thick ventricular septum and expresses doubt as to its permeability. Uses term "mitral" for left auriculo-ventricular valve. Gives diagnosis between thoracic and abdominal aortic aneurysms. This valuable copy exhibited was given to the Academy by Sir William Osler.

FABRICIUS AB AQUAPENDENTE, HIERONYMUS (1537-1619).
De Venarum Ostiolis.
Padua, L. Pasquati, 1603. Bound with his Opera anatomica, Padua, Meglietti, 1625.
1603 edition exhibited to show drawing of valves in the veins (Tabula Prima). These had earlier been discovered by Canano and Estienne (1551) but first delineated by Fabricius in 1574.

LOWER, RICHARD (1631-1691).
Tractatus de Corde item de Motu et Colore Sanguinis et Chyli in eum Transitu . . .
Amsterdam, D. Elzevir, 1669.
Described the structure of the heart more accurately than ever before. Pointed out the heart was a muscle. Described the arch of the aorta. (Drawings of this exhibited). Showed by insufflation of lungs that the red color of the blood in the aorta depended upon the inspired air. The copy exhibited was kindly lent by Dr. E. Moschcowitz, to show an early edition. The Academy also has a copy of this. "The London ed. of this year is usually quoted as the first." (*Bibl. Osler.*).

MALPIGHI, MARCELLO (1628-1694).
Opera Omnia.
Leyden, P. Vander Aa, 1687. 2 v. in 1.
Note *De Pulmonibus Epistola* II, p. 327, for microscopic demonstration of the capillary circulation in the frog's mesentery. Opened at drawings opposite p. 331.

THEBESIUS, ADAM CHRISTIANUS (1686-1732).
De Circulo Sanguinis in Corde.
Leyden, Potuliet, 1740.
Description (1708) of the coronary circulation including the vessels named after the author. Opened to show figures at the end of Book 3.

HENLE, FRIEDRICH GUSTAV JACOB (1809-1885).
Allgemeine Anatomie.
Leipzig, Voss, 1841.
Description of arterial endothelium, illustrated by drawing in Figure 3, Plate 1 at end of volume.

REMAK, ROBERT (1815-1865).
Ueber den Bau des Herzens.
Arch. f. Anat., Physiol. u. wissensch. Med., p. 76-78, 1850.
Description of the layers of muscle fibres in the heart and of its intrinsic ganglia.

MACCALLUM, JOHN BRUCE (1876-1906).
On the Muscular Architecture and Growth of the Ventricles of the Heart. In: Contributions to the Science of Medicine, dedicated . . . to William Henry Welch . . . p. 307-335.
Baltimore, Johns Hopkins Press, 1900.
Opened at p. 328 to show figure of 8 structure of fibres in heart muscle.

GROSS, LOUIS (1895-).
The Blood Supply to the Heart.
New York, Hoeber, 1921.
The classical work on this subject.

CONDUCTION SYSTEM.

KENT, ALBERT FRANK STANLEY (1863-).
Researches on the Structure and Function of the Mammalian Heart.
J. Physiol., XIV: 233-254, 1893.
Description of the auriculo-ventricular bundle in mammals. Opened at figure on p. 244.

HIS, WILHELM, JR. (1863-).
Die Thätigkeit des embryonalen Herzens und deren Bedeutung für die Lehre von der Herzbewegung beim Erwachsenen.
Arbeiten aus der Medizinischen Klinik zu Leipzig, 1893. p. 14-49. Leipzig, Vogel, 1893.
Description of the auriculo-ventricular bundle in the human embryo and other mammals. Opened at drawings on p. 22-23. Note also description at top of p. 23. This volume is a notable one in cardiovascular bibliography.

KEITH, (SIR) ARTHUR (1866-) and FLACK, MARTIN WILLIAM (1882-).
The Form and Nature of the Muscular Connections between the Primary Divisions of the Vertebrate Heart.
J. Anat. & Physiol., XLI: 172-189, 1906-1907.
Description of the sino-auricular node. Opened at figure on p. 181.

TAWARA, SUNAO ( ).

Das Reizleitungssystem des Säugetierherzens.

Jena, Fischer, 1906.

A complete account of the conduction fibres of the heart. Opened at Taf. VI.

PHYSIOLOGY.

SERVETUS VILLANOVANUS, MICHAEL (1509-1553).

De Restitutione Christianismi.

Nuremberg, Rau, 1790. Reprinted for Christoph Gottlieb von Murr, from the 1553 edition printed in Vienne, France, by Balthasar Arnollet.

Opened at p. 170. First accurate description of the lesser circulation. "1546. Earliest known form of the passage on the circulation by S. Represented by the MS., not in handwriting of Servetus, now in the Bibliothèque Nationale, Paris . . ." (Leonard L. Mackall, Servetus Notes, reprinted from Contributions to Medical and Biological Research, Dédicated to Sir William Osler . . . New York, Hoeber, 1919.) Only 3 copies of the original 1553 edition remain.

CAESALPINUS, ANDREAS (1520-1603).

Peripateticarum Quaestionum Libri Quinque.

Venice, Juntas, 1571.

Card exhibited gives reference to the denial by Caesalpinus that the vena cava originates from the liver. (Original not seen).

Quaestiones Medicae.

Venice, Juntas, 1593.

Card exhibited gives reference to the proof by Caesalpinus that veins carry blood *to* and *not from* the heart, made by demonstrating swelling of extremity distal to a ligature of a vein. (Original not seen).

HARVEY, WILLIAM (1578-1657).

Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus.

Frankfort, G. Fitzer, 1628.

Rare first edition bequeathed to the Academy by Dr. Walter James.

Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus, with English Translation and Annotations, by Chauncey D. Leake.

Springfield and Baltimore, Thomas, 1928.

Tercentennial edition.

An Anatomical Dissertation upon the Movement of the Heart and Blood in Animals being a Statement of the Discovery of the Circulation of the Blood. Facsimile Reproduction with Translation and Memoir.

Canterbury, G. Moreton, 1894.

Prelectiones Anatomiae Universalis. Edited with an Autotype Reproduction of the Original by a Committee of the Royal College of Physicians of London.

London, Churchill, 1886.

This volume was kindly lent for the exhibit by Dr. E. Moschcowitz. The Academy also has a copy.

WILD, F. ( ) (Ludwig's Laboratory).
Ueber die peristaltische Bewegung des Oesophagus, nebst einigen Bemerkungen über diejenigen des Darms.
Ztschr. f. rat. Med., V: 76-132, 1846.
Opened at p. 77 for first perfusion of the isolated heart.

RINGER, SYDNEY (1834-1910) and BUXTON, DUDLEY WILMOT ( ).
Concerning the Action of Small Quantities of Calcium, Sodium and Potassium Salts upon the Vitality and Function of Contractile Tissue and the Cuticular Cells of Fishes.
J. Physiol., VI: 154-161, 1885. See also vols. III and IV of this journal.
Ringer perfected Ludwig's method of keeping extravital tissues alive by showing the importance of calcium, sodium and potassium for muscular contraction. Ringer's solution.

LUDWIG, CARL FRIEDERICH WILHELM (1816-1895).
Lehrbuch der Physiologie des Menschen.
Leipzig & Heidelberg, Winter, 1852-1856. 2 v. See also Arch. f. Anat., Physiol. u. wissensch. Med., VI: 242-302, 1847.
Volume II opened at p. 85 to show illustration of float and kymograph for recording graphically the results of physiological experiments.

MAREY, ÉTIENNE JULES (1830-1904).
Physiologie médicale de la circulation du sang.
Paris, Delahaye, 1863.
Opened at page 54 to show Marey's tambours.

VOLKMANN, ALFRED WILHELM (1800-1877).
Die Hämodynamik.
Leipzig, Breitkopf & Härtel, 1850.
Opened at p. 138. First attempt at direct measurement of blood velocity by a U-shaped glass tube filled with salt solution, introduced into an artery. Improved by Ludwig in his stromuhr.

DOGIEL, J. (Ludwig's Laboratory).
Die Ausmessung der strömenden Blutvolumina.
Ber. ü. d. Verhandl. der K. Sächs. Gesellsch. der Wissensch. zu Leipzig, Math.-Phys. Classe, XIX: 200-275, 1867.
Measurement of blood velocity by the stromuhr. Opened at illustration on p. 202.

REIN, HERMANN ( ).
Die Thermo-Stromuhr.
Ztschr. f. Biol., LXXXVII: 394-418, 1928.
Measurement of the velocity of the blood stream by thermo-electric method.

TRAUBE, LUDWIG (1818-1876).
Ueber periodische Thätigkeits-Aeusserungen des vasomotorischen und Hemmungs-Nervencentrums.

Centralbl. f. d. med. Wissensch III: 881-885, 1865.

Traube-Hering Curves. Rhythmic variations in the tonus of the vaso-constrictor center.

BOWDITCH, HENRY PICKERING (1840-1911).

Uber die Eigenthümlichkeiten der Reizbarkeit, welche die Muskelfasern des Herzens zeigen.

Ber. ü. d. Verhandl. d. K. Sächs. Gesellsch. der Wissensch. zu Leipzig, Math.-Phys. Classe, XXIII: 652-689, 1871.

"All or none" property of cardiac contraction. Opened at p. 687, paragraph 2.

LUCIANI, LUIGI (1842-1919).

Eine periodische Function des isolirten Froschherzens.

Ber. ü. d. Verhandl. d. K. Sächs. Gesellsch. der Wissensch. zu Leipzig, Math.-Phys. Classe, XXV: 11-94, 1873.

"Treppe" phenomenon. Opened at figure on p. 19.

FRIEDENTHAL, HANS (1870-).

Ueber die Entfernung der extracardialen Herznerven bei Säugethieren.

Arch. f. Physiol., Leipzig, 1902, 135-145.

Proof of autonomy of heart muscle by elimination of the extracardiac nerves. Opened at p. 143, first paragraph.

KEITH, NORMAN MACDONNELL (1885-), ROWNTREE, LEONARD GEORGE (1883-) and GERAGHTY, JOHN T. ( ).

A Method for the Determination of Plasma and Blood Volume.

Reprinted from Arch. Int. Med., XVI: 547-576, 1915.

Use of vital red.

STARLING, ERNEST HENRY (1866-1927).

The Linacre Lecture on the Law of the Heart, given at Cambridge, 1915.

London, Longmans, Green, 1918.

Description of the heart-lung preparation and promulgation of the law of the heart. Opened at p. 6 to show diagram of apparatus and set-up.

GROLLMAN, ARTHUR (1901-).

The Determination of the Cardiac Output of Man by the Use of Acetylene.

Am. J. Physiol., LXXXVIII: 432-445, 1929.

BLOOD

HEWSON, WILLIAM (1739-1774).

Experimental Inquiries: Part the First, Containing an Inquiry into the Properties of the Blood with Remarks on Some of its Morbid Appearances.

London, J. Johnson, 1780.

See Chapter 1 for causes of coagulation of the blood.

HASSENFRATZ, M. ( ).

Mémoire sur la combination de l'oxigène avec le carbone & l'hydrogène

du sang, sur la dissolution de l'oxigène dans le sang, & sur la manière dont le calorique se dégage.

Ann. de chim., Paris IX: 261-274, 1791.

Internal respiration.

HOPPE-SEYLER, ERNST FELIX IMMANUEL (1825-1895).

Ueber das Verhalten des Blutfarbstoffes im Spectrum des Sonnenlichtes.

Virchow's Arch. f. path. Anat., XXIII: 446-456, 1862.

Spectroscopic studies of hemoglobin.

Ueber die chemischen und optischen Eigenschaften des Blutfarbstoffs.

Virchow's Arch. f. path. Anat., XXIX: 233-235, 1864.

HAMMARSTEN, OLOF (1841-).

Undersökningar af de s. k. Fibringeneratorerna Fibrinet Samt Fibrinogenets Koagulation.

Upsala Läkaref. förh., XI: 538-579, 1875-1876.

Fibrinogen and coagulation of the blood.

BOHR, CHRISTIAN (1855-1911), HASSELBACH, K. ( ) and KROGH, AUGUST.

Ueber einen in biologischer Beziehung wichtigen Einfluss, den die Kohlensäurespannung des Blutes auf dessen Sauerstoffbindung übt.

Skandinav. Arch. f. Physiol., XVI: 402-412, 1904.

Relation of carbon dioxide tension to oxygen combining power of the blood.

OSLER, (SIR) WILLIAM (1849-1919).

Bizzozero's New (?) Blood Element and Its Relation to Thrombus Formation. (See Centralbl. f. d. med. Wissensch., Jan. 14, 1882).

Med. News, XL: 250, 1882.

Description of blood-platelets. Osler had made a preliminary communication in 1873 (Centralbl. f. d. med. Wissensch., XI: 577-578, 1873.)

CARDIAC NERVES.

BEZOLD, ALBERT V. (1836-1868).

Untersuchungen über die Innervation des Herzens.

Leipzig, Engelmann, 1863.

I. Influence of the vagus on cardiac activity.

II. Influence of the cervical sympathetic on cardiac activity.

CYON, ÉLIE DE (1843-).

Les nerfs du coeur, anatomie et physiologie.

Paris, Alcan, 1905.

WEBER, ERNST HEINRICH (1795-1878) and WEBER, EDUARD FRIEDRICH (1806-1871).

Ann. Univ. di Med., Milano, 3 s., XX: 227, 1845.

Card exhibited gives reference to demonstration of inhibitory action of vagus nerve and cardiac standstill as a result of stimulation of the vagus endings. (Original not seen).

CAPILLARIES.

LOMBARD, WARREN PLIMPTON (1855-).

The Blood Pressure in the Arterioles, Capillaries and Small Veins of the Human Skin.

Am. J. Physiol., XXIX: 335-362, 1911-1912.

Use of the capillary microscope.

DALE, HENRY HALLETT (1875-) and RICHARDS, ALFRED NEWTON (1876-).

The Vasodilator Action of Histamine and of Some Other Substances.

J. Physiol., LII: 110-165, 1918-1919.

KROGH, AUGUST.

The Anatomy and Physiology of Capillaries.

New Haven, Yale University Press, 1929.

LEWIS, (SIR) THOMAS (1881-).

The Blood Vessels of the Human Skin and Their Responses.

London, Shaw & Sons, 1927.

BLOOD PRESSURE.

HALES, (REV.) STEPHEN (1677-1761).

Statical Essays, Containing Haemastaticks . . .

London, Innys and Manby, 1731-1733. 2 v. First edition.

Direct measurement of blood pressure by a pipe inserted into the femoral artery.

POISEUILLE, JEAN LÉONARD MARIE (1799-1869).

Recherches sur la force du coeur aortique.

Paris, Didot le Jeune, 1828. Thesis No. 166.

Use of the U-shaped tube with mercury for direct blood pressure measurement instead of the straight tube of Hales.

BASCH, SAMUEL SIEGFRIED KARL (1837-1905).

Der Sphygmomanometer und seine Verwerthung in der Praxis.

Reprinted from Berl. klin. Wchnschr., XXIV: 179-183; 206-208; 224-226; 244-246; 285-286, 1887.

Development of a small clinical sphygmometer based on Vierordt's idea of measuring blood pressure without opening the artery, by applying weights over the artery until pulsation ceased.

MARTIN, (SIR) CHARLES JAMES (1866-).

The Determination of Arterial Blood Pressure in Clinical Practice.

Brit. M. J., I: 865-870, 1905.

Improvement of Riva-Rocci's sphygmomanometer based on Marey's instrument (1876) which constricted the whole limb until pulsation ceased instead of pressing upon a limited portion of the artery.

FELLNER, BRUNO, JR. ( ).

Neuerung zur Messung des systolischen und diastolischen Druckes.

Verhandl. d. Kong. f. innere Med., XXIV: 404-407, 1907.

Use of the stethescope in determining blood pressure, first suggested by Korotkow (1905) and first used clinically by Krylow.

KRIES, N. v. ( ).
Ueber den Druck in den Blutcapillaren der menschlichen Haut.
Arb. a. d. Physiol. Anst. zu Leipzig, X: 69-80, 1875.
Measurement of capillary pressure (in the human skin).
Opened at illustrations on p. 70.

JANEWAY, THEODORE CALDWELL (1829-1911).
The Clinical Study of Blood-Pressure.
New York and London, Appleton, 1904.
An early comprehensive account of the physiological, technical and clinical aspects of modern blood pressure measurements.

THE PULSE AND ITS IRREGULARITIES.

FLOYER, (SIR) JOHN (1649-1734).
The Physician's Pulse-Watch.
London, Smith & Walford, 1707-1710. 2 v.
The first attempt to revive an exact timing of the pulse after Galileo and Sanctorius (pulsilogium).

PARRY, CALEB HILLIER (1755-1822).
An Experimental Inquiry into the Nature, Cause and Varieties of the Arterial Pulse . . .
London, Underwood, 1816.
An important original contribution after Floyer's methods had fallen into disuse, and the significance of pulse study had been deprecated.

POTAIN, PIERRE CARL EDOUARD (1825-1900).
Des mouvements et des bruits qui se passent dans les veines jugulaires.
Bull. et Mem. Soc. méd. d. Hôp. de Paris, 2nd series, IV: Mémoires, 3-27, 1867.
Venous pulse, and murmurs in the jugular vein.

MACKENZIE, (SIR) JAMES (1853-1925).
The Study of the Pulse.
Edinburgh & London, Pentland, 1902.
Use of the clinical polygraph. Opened at figures on p. 13.

WENCKEBACH, KARL FRIEDRICH (1864-).
Die unregelmässige Herztätigkeit und ihre klinische Bedeutung.
Leipzig & Berlin, Engelmann, 1914.
Classical studies in cardiac irregularities. Opened at figures between p. 82-83. (Taf. I).

TRAUBE, LUDWIG (1818-1876).
Ein Fall von Pulsus bigeminus nebst Bemerkungen über die Leberschwellungen bei Klappenfehlern und über acute Leberatrophie.
Ges. Beitr. z. Path. u. Physiol., III: 47-64, 1878.
Earliest description of cases with pulsus bigeminus. Figure on p. 49 shows radial curve in a case of pulsus alternans (first mention) which Traube considers a variation of pulsus bigeminus.

HOFFA, M. ( ) and LUDWIG, CARL FRIEDERICH WILHELM (1816-1895).
Einige neue Versuche über Herzbewegung.
Ztschr. f. rat. Med., IX: 107-144, 1850.
Experimental production of ventricular fibrillation.
Opened at description on p. 129.

MACWILLIAM, JOHN ALEXANDER (1857-).
Cardiac Failure and Sudden Death.
Brit. M. J., I: 6-8, 1889.
Ventricular fibrillation suggested as a cause of sudden death.
Fibrillar Contraction of the Heart.
J. Physiol., VIII: 296-310, 1887.
First description of auricular flutter in mammals.

RITCHIE, WILLIAM THOMAS (1873-).
Auricular Flutter.
New York, Hoeber, 1914.
First clinical recognition of auricular flutter in man. (Proc. Roy. Soc. Edinb., XXV: 1085, 1905). Opened at illustrations on p. 42 and opposite.

CUSHNY, ARTHUR ROBERTSON (1866-) and EDMUNDS, CHARLES WALLIS (1873-).
Paroxysmal Irregularity of the Heart and Auricular Fibrillation.
Studies in Pathology, Aberdeen University Series, No. 21, 95-110, 1906.
First recognition in man of auricular fibrillation, ascribed by the authors to vagal inhibition.

ROTHBERGER, CHARLES JULIUS ( ) and WINTERBERG, HEINRICH ( ).
Vorhofflimmern und Arhythmia perpetua.
Wien. klin. Wchnschr., XXII: 839-844, 1909.
First proof by means of the electrocardiograph that the completely irregular pulse was due to fibrillation of the auricle. Opened to show curves on p. 841.

LEWIS, (SIR) THOMAS (1881-).
Auricular Fibrillation and its Relationship to Clinical Irregularity of the Heart.
Heart, I: 306-372, 1909-1910.
Independent electrocardiographic demonstration that the completely irregular pulse was due to auricular fibrillation. (See Rothberger and Winterberg.) Opened to show figure on p. 355.

ELECTROCARDIOGRAPH

KOELLIKER, RUDOLPH ALBERT (1817-1905) and MÜLLER, HEINRICH.
Nachweis der negativen Schwankung des Muskelstroms am natürlich sich contrahirenden Muskel.
Verhandl d. Phys.-Med. Gesellsch. zu Würzburg, VI: 528-533, 1856.
Action currents due to cardiac contraction produce galvanic reaction in sciatic nerve preparation. (See p. 533.)

SANDERSON, (SIR) JOHN SCOTT BURDON (1828-1905) and PAGE, F. J. M.
On the Time-relations of the Excitatory Process in the Ventricle of the Heart of the Frog.
J. Physiol., II: 384-435, 1879-1880.
Investigation with the capillary electrometer of the electric currents produced by cardiac contraction, previously recognized by Matteucci (1843) and by Kölliker and Müller (1856). Opened to show illustration opposite p. 435.

EINTHOVEN, WILLEM (1860-1927).
Die galvanometrische Registrirung des menschlichen Elektrokardiogramms, zugleich eine Beurtheilung der Anwendung des Capillar-Elektrometers in der Physiologie.
Arch. f. ges. Physiol., XCIX: 472-480, 1903.
First description of the string galvanometer for registration of the human electrocardiogram. Opened at p. 472-473.

WALLER, AUGUSTUS DÉSIRÉ (1856-1922).
The Electrical Action of the Human Heart.
London, University of London Press, 1922.
In the *Phil. Tr.*, 1889, London, CLXXX B: 169-194, 1890, Waller demonstrated the possibility of obtaining curves of the action currents of cardiac contraction without opening the chest, by photographing the mercury meniscus of the capillary electrometer, after leading off from the extremities.

EXPERIMENTAL AND CLINICAL HEART BLOCK.

STANNIUS, HERMANN (1808-1883).
Versuche am Froschherzen.
Arch. f. Anat., Physiol. u. wissensch. Med., 85-93, 1852.
Complete heart block, first produced in frog by "Stannius ligature" between auricle and ventricle (see p. 87, paragraph 9). Physiologic heart block had already been noted by Harvey (De Motu Cordis, Chapter iv).

GASKELL, WALTER HOLBROOK (1847-1914).
On the Innervation of the Heart, with Especial Reference to the Heart of the Tortoise.
J. Physiol., IV: 43-127, 1883.
Physiologic demonstration of the muscular continuity between the auricle and ventricle in reptiles. The Gaskell clamp. Opened to show p. 57.

CHEYNE, JOHN (1777-1836).
A Case of Apoplexy in which the Fleshy Part of the Heart was Converted into Fat.
Dublin Hosp. Rep., II: 216-223, 1818.
Association of the type of breathing now known as "Cheyne-Stokes" with fatty degeneration of the heart. Opened to show p. 22. (See last paragraph).

ADAMS, ROBERT (1791-1875).

Cases of Diseases of the Heart, accompanied with Pathological Observations.

Dublin Hosp. Rep., IV: 353-453, 1827.

Description of two cases (See p. 390 and 396) of convulsions in heart disease, one associated with irregular breathing and slow pulse. Ascribed to fatty degeneration of the heart. Later termed by Huchard, "Maladie de Stokes-Adams."

STOKES, WILLIAM (1804-1878).

Observations on Some Cases of Permanently Slow Pulse.

Dublin J. M. Sc., II: 73-85, 1846.

Cases of Stokes-Adams disease including also a description (p. 83) of what is now termed Cheyne-Stokes respiration.

EPPINGER, HANS ( ), and ROTHBERGER, J. ( ).

Ueber die Folgen der Durchschneidung der Tawaraschen Schenkel des Reizleitungssystems.

Ztschr. f. klin. Med., LXX: 1-20, 1910.

Experimental production of bundle branch block. Opened to show illustrations p. 6-7.

PATHOLOGY.

BONET, THEOPHILE (1620-1689).

Sepulchretum sive Anatomia Practica ex Cadaveribus Morbo Denatis.

Geneva, L. Chouët, 1679. 2 v.

First edition. Early observations on palpitation and cardiac pain associated with a variety of pathologic findings such as aortic aneurysm (page 496 at which volume I was opened) and numerous other thoracic affections.

LANCISI, GIOVANNI MARIA (1654-1720).

De Subitaneis Mortibus.

Venice, A. Poleti, 1708.

Description of aneurysm as a cause of sudden death. Opened at p. 116-117.

De Motu Cordis et Aneurysmatibus.

Leyden, Bonk & Steenman, 1740.

Detailed description of aneurysm of the heart with thinning of the walls (=dilatation) and, distinguished from this, aneurysm with thickening (=excentric hypertrophy). Associated swelling of the jugular veins with dilatation of the right ventricle.

MORGAGNI, GIOVANNI BATTISTA (1682-1771).

De Sedibus et Causis Morborum per Anatomen Indagatis.

Venice, Remondiniana, 1761. 2 v. in 1.

Contains a full description of valvular and muscular lesions of the heart. Morgagni attributed cyanosis to venous stasis; recognized and distinguished anatomically cardiac hypertrophy and dilatation; and realized the luetic origin of aortic aneurysm. First described case of

angina pectoris with dilated and calcified aorta, as well as a case of rupture of the heart. Opened at p. 70, paragraph 7, describing the first case reported of what is now known as Stokes-Adams disease.

BAILLIE, MATTHEW (1760-1823).

The Works of . . .

London, Longman Hurst . . . 1825. 2 v.

Vol. II, The Morbid Anatomy of Some of the Most Important Parts of the Human Body. Note p. 27 for ossification of the coronary arteries, p. 31 for excrescences on the heart valves and for rupture of the heart.

VIEUSSENS, RAYMOND (1641-1715).

Novum Vasorum Corporis Humani Systema.

Amsterdam, Marret, 1705.

First Edition. Early contributions to heart disease. Clinical and pathological descriptions of mitral stenosis, aortic stenosis and aortic insufficiency, with remarkable description of the pulse in the latter disease.

PHYSICAL DIAGNOSIS.

AUENBRUGGER, LEOPOLD (1722-1809).

Inventum Novum ex Percussione Thoracis Humani . . .

Vienna, Trattner, 1761.

First edition. First introduction of immediate percussion. Neglected until rescued from oblivion by Corvisart's translation, 1808, just before the death of Auenbrugger.

This volume was kindly lent to the Academy for the exhibit by Dr. E. Moschcowitz.

LAENNEC, RÉNÉ-THÉOPHILE-HYACINTHE (1781-1826).

De l'auscultation médiate . . .

Paris, Brosson & Chaudé, 1819. 2 v.

First edition. Description of the stethoscope and mediate auscultation. Unlike the discovery of percussion, interest in auscultation followed rapidly and was widespread, so that though Laennec died at 45 of phthisis, he saw his discoveries accepted everywhere.

THERAPY.

WITHERING, WILLIAM (1741-1799).

An Account of the Foxglove and Some of its Medical Uses.

Birmingham, Robinson, 1785.

Use of digitalis for certain forms of dropsy. 1785. A colored plate of the foxglove is shown.

CLINICAL HEART DISEASE.

DE SENAC, JEAN BAPTISTE (1693-1770).

Traité de la structure du coeur, de son action, et de ses maladies.

Paris, Barbou, 1777. 2 v.

First edition, 1749. First textbook on the heart alone. Contains a comprehensive account of cardiac diseases. Called an "opus magni sudoris" because of a lifetime devoted to its study. Most important is his emphasis on the role of inflammation in heart disease. Beautiful illustrations at conclusion of volume 1 were exhibited.

This copy was kindly lent for the exhibit by Dr. Emanuel Libman of New York, and both volumes of the work have since been presented by him to the New York Academy of Medicine.

CORVISART, JEAN NICOLAS (1755-1821).

Essai sur les maladies et les lésions organiques du coeur et des gros vaisseaux.

Paris, Migneret, 1806.

First edition. Laid great stress on the frequency of heart disease. Corvisart was the first to refer to thrills. By translating Auenbrugger's work on percussion, he saved it from oblivion. Emphasized importance of the myocardium, and was one of the first to describe mitral stenosis.

MYOCARDIAL DISEASE.

FRANK, JOANNIS PETRUS (1745-1821).

De Curandis Hominum Morbis . . .

Vienna, A. Doll, 1810. 7 v.

Volume II opened to show p. 114. Use of the term "carditis" for inflammatory disease of the heart. First used by Aurelianus (see de Senac).

SOBERNHEIM, JOSEPH FRIEDRICH (1804-1846).

Akute idiopathische Herzentzündung. In his: Praktische Diagnostik der innern Krankheiten, p. 118-120.

Berlin, Hirschwald, 1837.

First use of the term "myocarditis."

LEYDEN, ERNST v. (1832-1910).

Ueber Fettherz.

Ztschr. f. klin. Med., V: 1-25, 1882.

ROY, JOHN GEORGE ( ), and ADAMI, J. G. ( ).

Remarks on Failure of the Heart from Overstrain.

Brit. M. J., II: 1321-1326, 1888.

KREHL, LUDOLF v. (1861-).

Die Erkrankungen des Herzmuskels und die nervösen Herzkrankheiten.

Vienna & Leipzig, Hölder, 1913.

ALBRECHT, EHRENFRIED (1864-).

Der Herzmuskel und seine Bedeutung für Physiologie, Pathologie und Klinik des Herzens.

Berlin, Springer, 1903.

ANGINA PECTORIS AND CORONARY ARTERY DISEASE.

HEBERDEN, WILLIAM (1710-1801).

Some Account of a Disorder of the Breast.

Med. Tr. Roy. Coll. Phys., 2. ed., London, II: 59-67, 1786.

First description, 1768, of angina pectoris based on the observation of 20 cases. Heberden originated the term.

PARRY, CALEB HILLIER (1755-1822).

An Inquiry into the Symptoms and Causes of the Syncope Anginosa Commonly Called Angina Pectoris.

London, Cadell & Davies, 1799.

Concluded there was an important connection between ossification or obstruction of the coronary arteries and angina pectoris. Credited Jenner with discovery of this. Opened at p. 110.

BURNS, ALLAN (1781-1813).

Observations on Some of the Most Frequent and Important Diseases of the Heart . . .

Edinburgh, Bryce, 1809.

First English treatise on diseases of the heart. The first formal text-book was that of Hope, 1839. Burns first ascribed the causation of pain in "angina pectoris" to anemia of the heart muscle as a result of coronary obstruction (p. 138).

NOTHNAGEL, CARL WILHELM HERMANN (1841-1905).

Angina Pectoris Vasomotoria.

Deutsches Arch. f. klin. Med., III: 309-322, 1867.

BRUNTON, (SIR) THOMAS LAUDER (1844-1916).

On the Use of Nitrite of Amyl in Angina Pectoris.

Lancet, II: 97-98, 1867.

HAMMER, ADAM (1818-1878).

Ein Fall von thrombotischem Verschlusse einer der Kranzarterien des Herzens.

Wien. med. Wchnschr., XXVIII: 97-102, 1878.

First case of coronary thrombosis with correct diagnosis ante mortem.

LEYDON, ERNST v. (1832-1910).

Ueber die Sclerose der Coronar-Arterien und die davon abhängigen Krankheitszustände.

Ztschr. f. klin. Med., VII: 459-486, 539-580, 1884.

Discussion of cases of acute and chronic coronary occlusion.

OBRASTZOW, W. P. and STRASCHESKO, N. D.

Zur Kenntnis der Thrombose der Koronararterien des Herzens.

Ztschr. f. klin. Med., LXXI: 116-132, 1910.

First thorough description of coronary thrombosis including 5 cases, 2 of which had been diagnosed during life. Prior to that, Hammer, 1878, had correctly made such a diagnosis ante mortem.

HERRICK, JAMES BRYAN (1861-).
Clinical Features of Sudden Obstruction of the Coronary Arteries.
Reprinted from J. A. M. A., LIX: 2015-2020, 1912.

CHIRAC, PIERRE (1650-1732). Quoted by Dimitri Pletnew in:
Störungen der Synergie beider Herzkammern.
Ergebn. d. inn. Med. u. Kinderh., III: 429-446, 1909.
Quotation is on p. 434. Pletnew states that Chirac was the first to attempt ligation of the coronary arteries, in the latter part of the 17th century.

SMITH, FRED M.
The Ligation of Coronary Arteries with Electrocardiographic Study.
Arch. Int. Med., XXII: 8-27, 1918.

PARDEE, HAROLD ENSIGN BENNETT (1886).
An Electrocardiographic Sign of Coronary Artery Obstruction.
Reprinted from Arch. Int. Med., XXVI: 244-257, 1920.

OPPENHEIMER, BERNARD SUTRO (1876-) and ROTHSCHILD, MARCUS ADOLPHUS (1887-).
Abnormalities in the QRS Group of the Electrocardiogram associated with Myocardial Involvement.
Reprinted from Proc. Soc. Exper. Biol. & Med., XIV: 57-59, 1916-17.

LIBMAN, EMANUEL (1872-) and SACKS, BENJAMIN (1896-).
A Case Illustrating the Leucocytosis of Progressive Myocardial Necrosis following Coronary Artery Thrombosis.
Reprinted from Am. Heart J., II: 321-326, 1927.

ALLBUTT, (SIR) THOMAS CLIFFORD (1836-1925).
Diseases of the Arteries Including Angina Pectoris.
London, Macmillan, 1915.
Note Vol. II, p. 245-248, for tobacco angina, and Vol. II, p. 415-416 and 449 for cause of pain in angina pectoris.

GALLAVARDIN, LOUIS (1875-).
Les angines de poitrine.
Paris, Masson, 1925.

DANIÉLOPOLU, D.
L'Angine de poitrine et l'angine abdominale.
Paris, Masson, 1927.
Note surgical treatment.

LEVINE, SAMUEL ALBERT (1891-).
Coronary Thrombosis: its Various Clinical Features.
Medicine Monographs, XVI. Baltimore, Williams & Wilkins, 1929.

CONGENITAL HEART DISEASE.

PEACOCK, THOMAS BEVILL (1812-1882).
On Malformations . . . of the Human Heart with Original Cases.
London, Churchill, 1858.

Congenital cardiac malformations had first been observed by de Senac [e. g. absence of interventricular septum (1749)] and Morgagni [pulmonary stenosis (1761)].

ROKITANSKY, CARL (FR.) VON (1804-1878).
Die Defecte der Scheidewände des Herzens.
Vienna, Braumüller, 1875.
Description of septal defects in congenital heart disease which he believed were due to fetal endocarditis. Beautiful illustrations. Opened at figures on p. 138-139.

ABBOTT, MAUDE ELIZABETH SEYMOUR (1869-).
Congenital Cardiac Disease. In: Osler's Modern Medicine.
Philadelphia, Lea & Febiger, 1927. Vol. IV, p. 612-812.
Most comprehensive description of our present knowledge of congenital heart disease.

ENDOCARDITIS AND VALVULAR DISEASE.

WELLS, WILLIAM CHARLES (1757-1817).
On Rheumatism of the Heart.
Tr. Soc. Improve. M. & Chir. Knowl., London, III: 373-424, 1812.
Description of 14 cases of rheumatism of the heart, for recognition of which credit is given to David Pitcairn (1788).

BOUILLAUD, JEAN BAPTISTE (1796-1881).
New Researches on Acute Articular Rheumatism in General; and Especially on the Law of Coincidence of Pericarditis and Endocarditis with this Disease. (Translated from the French ed. of 1836 by J. Kitchen).
Philadelphia, Haswell, Barrington & Haswell, 1837.
The rheumatic etiology of certain forms of cardiac disease first recognized by David Pitcairn (1749-1809) and later by others (Jenner, Corvisart, Hope) was definitely demonstrated by clinico-pathologic correlation by Bouillaud, who also recognized the disease, and originated the term, "acute endocarditis." Endocarditis had been previously described by Corvisart, 1806, and Andral, 1829.

HODGKIN, THOMAS (1798-1866).
On Retroversion of the Valves of the Aorta.
London M. Gaz., III: 433-443, 1829.
Concise pathologic and clinical description of cases with aortic insufficiency. Opened at descriptions on p. 440-441.

CORRIGAN, (SIR) DOMINIC JOHN (1802-1880).
On Permanent Patency of the Mouth of the Aorta, or Inadequacy of the Aortic Valves.
Edinburgh M. & S. J., XXXVII: 225-245, 1832.
Complete description of aortic insufficiency including the character of the pulse which bears the author's name. The valve lesion had been described earlier by William Cooper (1703) and the disease picture, in-

cluding the character of the pulse, by Vieussens (1715). Opened at figures opposite p. 225 to illustrate the character of the lesion which Hodgkin so aptly termed retroversion of the aortic valves.

KIRKES, WILLIAM SENHOUSE (1823-1864).
On Some of the Principal Effects Resulting from the Detachment of Fibrinous Deposits from the Interior of the Heart, and Their Mixture with the Circulating Blood.
Med.-Chir. Tr., London, XXXV: 281-324, 1852.
First description of bacterial endocarditis with emboli from valvular vegetations.

WILKS, (SIR) SAMUEL (1824-1911).
Capillary Embolism or Arterial Pyemia.
Guy's Hosp. Rep., 3rd series, XV: 29-35, 1870.
Emphasis on the frequency of arterial pyemia in association with chronic heart disease. Opened at p. 35.

HEIBERG, HJALMAR (1837-1897).
Ein Fall von Endocarditis ulcerosa puerperalis mit Pilzbildungen im Herzen (Mycosis Endocardii).
Virchow's Arch. f. path. Anat., LVI: 407-424, 1872.

OSLER, (SIR) WILLIAM (1849-1919).
The Gulstonian Lectures on Malignant Endocarditis.
London, 1885. Delivered at the Royal College of Physicians.
First graphic clinical and pathologic picture of bacterial endocarditis based on over 200 cases.

HORDER, (SIR) THOMAS JEEVES (1871-).
Infective Endocarditis with an Analysis of 150 Cases and with Special Reference to the Chronic Form of the Disease.
Quart. J. Med., II: 289-324, 1908-1909.

LIBMAN, EMANUEL (1872-) and CELLER, H. L. ( ).
The Etiology of Subacute Infective Endocarditis.
Am. J. M. Sc., CXL: 516-527, 1910.

LIBMAN, EMANUEL (1872-).
A Study of the Endocardial Lesions of Subacute Bacterial Endocarditis.
Reprinted from Am. J. M. Sc., CXLIV: 313-327, 1912.
Clinical and pathologic picture of subacute bacterial endocarditis in the bacteria-free (healing and healed) stage.

GERHARDT, DIETRICH (1866-1921).
Die Endokarditis.
Vienna & Leipzig, Hölder, 1914.

BAEHR, GEORGE (1887-).
Glomerular Lesions of Subacute Bacterial Endocarditis.
Am. J. M. Sc., CXLIV: 327-329, 1912.

LIBMAN, EMANUEL (1872-)

Characterization of Various Forms of Endocarditis.

J. A. M. A., LXXX: 813-818, 1923.

DISEASES OF VESSELS—ANEURYSM, THROMBOSIS, EMBOLISM.

HOME, (SIR) EVERARD (1763-1832).

An Account of Mr. Hunter's Method of Performing the Operation for the Cure of the Popliteal Aneurism.

Tr. Soc. Improve. M. & Chir. Knowl., London, I: 138-181, 1793.

Additional Cases to illustrate Mr. Hunter's Method of Performing the Operation for the Cure of the Popliteal Aneurism.

Tr. Soc. Improve. M. & Chir. Knowl., London, II: 235-256, 1800.

HODGSON, JOSEPH (1788-1869).

A Treatise on the Diseases of Arteries and Veins, Containing the Pathology and Treatment of Aneurisms and Wounded Arteries.

London, Underwood, 1815.

LOBSTEIN, JEAN-GEORGES-CHRÉTIEN-FRÉDÉRIC-MARTIN (1777-1835).

Von der Verdickung der Arterien; oder der Arteriosklerose. In his: Lehrbuch der pathologischen Anatomie (Translated from the French edition of 1829-1833 by Neurohr), II: Chapter vi, 472-474.

Stuttgart, Fr. Brodhag, 1834-1835. 2 v.

First use of the term arteriosclerosis.

VIRCHOW, RUDOLF LUDWIG KARL (1821-1902).

Ueber die akute Entzündung der Arterien.

Virchow's Arch. f. path. Anat., I: 272-404, 1847.

Virchow firmly established the existence of acute arteritis (acute periarteritis and mesaortitis).

KUSSMAUL, ADOLF (1822-1902) and MAIER, RUDOLF (1824-1888).

Ueber eine bisher nicht beschriebene eigenthümliche Arterienerkrankung (Periarteritis nodosa), die mit Morbus Brightii und rapid fortschreitender allgemeiner Muskellähmung einhergeht.

Deutsches Arch. f. klin. Med., I: 484-518, 1865-1866.

First description of periarteritis nodosa associated with Bright's disease.

FELTZ, VICTOR TIMOTHÉE (1835-1893).

Traité clinique et expérimental des embolies capillaires.

Paris, Baillière, 1870.

GULL, (SIR) WILLIAM WITHEY (1816-1899) and SUTTON, HENRY GOWEN (1837?-1891).

On the Pathology of the Morbid State Commonly called Chronic Bright's Disease with Contracted Kidney ("Arterio-capillary Fibrosis").

Med.-Chir. Tr., London, LV: 273-326, 1872.

EPPINGER, HANS (1846-1916).

Pathogenesis (Histogenesis und Aetiologie) der Aneurysmen einschliesslich des Aneurysma Equi verminosum.

Arch. f. klin. Chir., XXXV: 1887.

RAYNAUD, A. G. MAURICE (1834-1881).
On Local Asphyxia and Symmetrical Gangrene of the Extremities. (Translated from the French ed. of 1862 by Thomas Barlow.)
Selected Monographs of the New Sydenham Soc., CXXI: 1-199, 1888.

WELCH, WILLIAM HENRY (1850-).
Thrombosis. In: A System of Medicine, ed. by Sir Clifford Allbutt & Sir Humphry D. Rolleston. Volume VI, p. 691-762.
London, Macmillan, 1909.
Embolism. Ibid., p. 762-821.

LJUNGDAHL, MALTE ( ).
Untersuchungen über die Arteriosklerose des kleinen Kreislaufs.
Wiesbaden, Bergmann, 1915.
Amplification of the study of pulmonary sclerosis probably first described by Corvisart.

BUERGER, LEO (1879-).
The Circulatory Disturbances of the Extremities.
Philadelphia & London, Saunders, 1924.
Description of Thromboangiitis Obliterans (Buerger's Disease).

ARRILLAGA, F. C. ( ).
La arteritis pulmonar y su cuadro clínico.
Buenos Aires, "El Ateneo," 1925.
Discussion of pulmonary sclerosis including Ayerza's disease.

MOSCHCOWITZ, ELI (1879-).
Hypertension of the Pulmonary Circulation.
Am. J. M. Sc., CLXXIV: 388-406, 1927.
The Cause of Arteriosclerosis.
Reprinted from Am. J. M. Sc., CLXXVIII: 244-267, 1929.
Pseudo, or Transient, Arteriosclerosis.
J. A. M. A., XC: 1526-1527, 1928.

DUCUING, J. ( ).
Phlebites thromboses et embolies post-opératoires.
Paris, Masson, 1929.

REFERENCE BOOKS.

Microscopic Anatomy.

MOELLENDORFF, WILHELM v. (1887-).
Handbuch der mikroskopischen Anatomie des Menschen. Vol. VI, Part 1.
Berlin, Springer, 1930.

Anatomy.

TANDLER, JULIUS (1869-).
Anatomie des Herzens. In: Bardeleben's Handbuch der Anatomie des Menschen, Vol. III, Part 1.
Jena, Fischer, 1913.

Physiology.

Handbuch der normalen und pathologischen Physiologie (Bethe, v. Bergmann, Embden, Ellinger), Vol. VII.
Berlin, Springer, 1926-1927.

Hess, Walter R. ( ).
Die Regulierung des Blutkreislaufes.
Leipzig, Thieme, 1930.

Pathology.

Henke, E. Friedrich K. (1868-) and Lubarsch, Otto (1860-).
Handbuch der speziellen pathologischen Anatomie und Histologie. Vol. II.
Berlin, Springer, 1924.

X-ray

Assmann, Herbert ( ).
Die klinische Röntgendiagnostik der inneren Erkrankungen. Part 1.
Leipzig, Vogel, 1928. 2 v.

Koch, Walter (1880-), and Wieck, Wilhelm ( ).
Anatomische Analyse des Röntgenbildschattens des Herzens und der Interlobärspalten der Lungen.
Jena, Fischer, 1930.

Electrocardiography.

Lewis, (Sir) Thomas (1881-).
The Mechanism and Graphic Registration of the Heart Beat.
London, Shaw, 1925.

Wenckebach, Karl Friedrich ( ), and Winterberg, Heinrich ( ).
Die Unregelmässige Herztätigkeit.
Leipzig, Engelmann, 1927. 2 v.

Symptomatology.

Laubry, Charles (1872-).
Leçons de sémiologie cardio-vasculaire, les troubles fonctionnels.
Paris, Doin, 1924.

Physical diagnosis.

Norris, George William and Landis, Henry Robert Murray (1875-).
Diseases of the Chest and the Principles of Physical Diagnosis.
Philadelphia and London, Saunders, 1929.

Heart disease.

White, Paul Dudley (1886-).
Heart Disease.
New York, Macmillan, 1931.

Vaquez, Henri (1860-).
Diseases of the Heart. (English translation by George Frederick Laidlaw).
Philadelphia and London, Saunders, 1924.

Clerc, M. A. ( ).
Coeur. In: Précis de pathologie médicale, Vol. IV, p. 1-815.
Paris, Masson, 1931.

Clerc, M. A. ( ), and Deschamps, Noël ( ).
Vaisseaux. In: Précis de pathologie médicale, Vol. IV, p. 819-1278.
Paris, Masson, 1931.

Romberg, Ernst ( ).
Lehrbuch der Krankheiten des Herzens und der Blutgefässe.
Stuttgart, Enke, 1925.

East, Charles Frederick Terence (1894-) and Bain, Cyril William Curtis (1895-).
Recent Advances in Cardiology.
Philadelphia, Blakiston, 1929.

TEXTBOOKS ON HEART DISEASE—ENGLISH.

Hope, James (1801-1841).
A Treatise on the Diseases of the Heart and Great Vessels.
London, Kidd, 1832.
First English textbook on heart disease. Note description of arteritis, p. 138 ff.; of valvular disease, p. 307-345; and emphasis on "asthma" resulting from disease of the heart, p. 345-365. Hope described the diastolic murmur in mitral stenosis and noted the "jerking pulse" in aortic insufficiency.

Stokes, William (1804-1878).
The Diseases of the Heart and the Aorta.
Dublin, Hodges & Smith, 1854.
Note p. 36 ff. for pericarditis, p. 126-127 for latent valvular disease, p. 276-277 for dilatation of the heart with or without hypertrophy, p. 139 ff. for extreme ossific disease of the aortic orifice.

Walshe, Walter Hayle (1812-1892).
A Practical Treatise on the Diseases of the Heart and Great Vessels, Including the Principles of Physical Diagnosis.
Philadelphia, Blanchard & Lea, 1862.
Note exocardial or pericardial murmurs, p. 95-101; cardiac paraesthesiae, p. 132-133; cardiac anemia, p. 168; calcification in the heart, p. 259-260.

Latham, Peter Mere (1789-1875).
The Collected Works of . . .
London, The New Sydenham Soc., 1876. 2 v.
See Lecture IX, Vol. I for inflammation of the lungs accompanying acute rheumatic fever, p. 96-108.

Sibson, Francis (1814-1786).
Collected Works . . . Edited by William M. Ord.
London, Macmillan, 1881. 4 v.
See Vols. III and IV, Chapters xiii and xvi for sections on pericarditis.

FOTHERGILL, JOHN MILNER (1841-1888).
The Heart and Its Diseases . . .
Philadelphia, Lindsay & Blakiston, 1879.
Note valvulitis, p. 154 ff., and the gouty heart, p. 400 ff.

PAUL, CONSTANTIN CHARLES THÉODOR (1833-1896).
Diagnosis and Treatment of Diseases of the Heart. (English Translation from 1883 French ed.).
New York, Wood, 1884.

BALFOUR, GEORGE WILLIAM (1823-1903).
Clinical Lectures on Diseases of the Heart and Aorta.
London, Churchill, 1882.
Note p. 208 ff. for murmurs in the pulmonary region, p. 234 for variation and disappearance of cardiac murmurs.

BRAMWELL, (SIR) BYROM.
Diseases of the Heart and Thoracic Aorta.
New York, Appleton, 1884.
Note Chapter iv, p. 296 ff. for pericarditis.

SANSOM, ARTHUR ERNEST (1839-1907).
The Diagnosis of Diseases of the Heart and Thoracic Aorta.
London, Griffin, 1892.
Note p. 83-87, for skin manifestations in rheumatism.

GIBSON, GEORGE ALEXANDER (1854-1913).
Diseases of the Heart and Aorta.
Edinburgh & London, Pentland, 1898.
Note adventitious murmurs, p. 153-155; cardiac murmurs, p. 155-174; auscultation of the veins, 205-206; extra cardiac symptoms in cardiac disease, p. 207-254; diseases of the pericardium, p. 313-392; endocarditis with infective lesions, 405-435; fatty degeneration, p. 656-664; and spontaneous rupture of the heart, p. 716-720.

BROADBENT, (SIR) WILLIAM HENRY (1835-1907) and BROADBENT, JOHN FRANCIS HARPIN.
Heart Disease and Aneurysm of the Aorta with Special Reference to Prognosis and Treatment.
New York, Wood, 1906.

STEELL, GRAHAM (1851-).
Textbook on Diseases of the Heart.
Philadelphia, Blakiston, 1906.

MACKENZIE, (SIR) JAMES (1853-1925).
Diseases of the Heart. (Oxford Medical Publications).
London, Frowde, 1918.
Note Chapter xiv, p. 76-87, for angina pectoris, and p. 168-276, for cardiac irregularities.

HIRSCHFELDER, ARTHUR DOUGLASS (1879-).
Diseases of the Heart and Aorta.
Philadelphia & London, Lippincott, 1918.

COWAN, JOHN (1870-) and RITCHIE, WILLIAM THOMAS ( ).
Diseases of the Heart.
London, Arnold, 1922.

PRICE, FREDERICK WILLIAM.
Diseases of the Heart, Their Diagnosis, Prognosis and Treatment by Modern Methods.
Milford, Oxford University Press, 1927.

REID, WILLIAM DUNCAN (1885-)
The Heart in Modern Practice.
Philadelphia & London, Lippincott, 1928.

CRUMMER, LEROY (1872-).
Clinical Features of Heart Disease.
New York, Hoeber, 1930.
Distinguished by its conciseness and lucidity of style.

TEXTBOOKS ON HEART DISEASE—FRENCH.

PIGEAUX, ANTOINE-LOUIS-JULES, (1807-).
Traité pratique des maladies du coeur contenant des recherches historiques, anatomiques et physiologiques spéciales sur cet organe.
Paris, Rouvier, 1839.
Note partial aneurysms of the heart, p. 491-501.

FRIEDREICH, NIKOLAUS (1825-1882).
Traité des maladies du coeur. (Translated from German 2nd edition of 1867).
Paris, Delahaye, 1873.
Note p. 344-577, for diseases of the endocardium.

PETER, MICHEL (1824-).
Traité clinique et pratique des maladies du coeur et de la crosse de l'aorte.
Paris, Baillière, 1883.
Note p. 83-84 for dry pericarditis in Bright's disease, p. 170-208 for acute myocarditis, p. 299 for pathogenesis of hypertrophy of the left ventricle.

PAUL, CONSTANTIN CHARLES THÉODOR (1833-1896).
Diagnostic et traitement des maladies du coeur.
Paris, Asselin & Houzeau, 1887.
Note p. 35 for displacements of the heart, p. 132-136 for endocarditis and anemia, Chapter xix, xx, for diseases of the arch of aorta including Hodgson's disease, p. 465-473 for chronic partial aneurysms, p. 487-500 for angina pectoris.
This volume was kindly lent for the exhibit by Dr. Libman.

DUROZIEZ, PAUL-LOUIS (1826-1897).
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Paris, Steinheil, 1891.
See p. 96-103 for presystolic click of the valves of the veins of the

neck, subclavicular and femoral regions; p. 109-110 for shifting of the apex; p. 201-210 for adhesive pericarditis; p. 225-227 for angiosclerosis; Chapter v. for insufficiencies and stenoses; p. 333-334 for the rheumatic heart—"Admitting the generalization of the disease, we admit the generalization of the cardiac lesion; it is not a single valve which is affected, it is all the valves; it is the whole endocardium, the pericardium, the myocardium; it is the arteries; it is the veins." See also p. 355-360 for lesions and vascular murmurs in the 2nd left intercostal space.

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See p. 155 ff. for acute rheumatic endocarditis; p. 363-506 for cardiopulmonary murmurs; p. 839-842 for rheumatic phlebitis.

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The most famous German classic on heart disease.

FRIEDREICH, NIKOLAUS (1825-1882).
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Note p. 293-328 for cardiac neuroses.

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Die Krankheiten des Herzens und ihre Behandlung.
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Note p. 259-263 for digestive reflex neurosis with cardiac manifestations.

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Mittenwald, Nemayer, [1931], 76 p.

Scott, S. G. Radiology in relation to medical jurisprudence.
London, Cassell, 1931, 65 p.

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München, Lehmann, 1932 [1931], 310 p.

Stewart, (Sir) J. P. The diagnosis of nervous diseases. 7. ed.
London, Arnold, 1931, 730 p.

Thompson, C. J. S. Poisons and poisoners.
London, Shaylor, [1931], 392 p.

Trier, G. Die Alkaloide; eine Monographie der natürlichen Basen. 2. Aufl.
Berlin, Borntraeger, 1931, 1061 p.

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N. Y., Century, [1931], 604 p.

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London, Kimpton, 1931, 321 p.

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A SELECTED LIST OF NEW PERIODICALS ADDED IN 1931

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Wir Zuckerkranken. (Deutsche diabetikerbunde E. V.)
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PROCEEDINGS OF ACADEMY MEETINGS
JANUARY, 1932

STATED MEETINGS

ANNUAL MEETING
Thursday Evening, January 7, at 8:30 o'clock

ORDER

I. EXECUTIVE SESSION
Reading of the Minutes
Election of Fellows
II. PRESENTATION OF ANNUAL REPORTS
The Council, John A. Hartwell
The Trustees, Eugene H. Pool
The Treasurer, Seth M. Milliken
Committees: (Read by title)
Admission, Alexis V. Moschcowitz
Library, Eugene F. DuBois
Public Health Relations, James Alexander Miller
Medical Education, Nellis B. Foster
Sections, Lewis A. Conner
Professional Standards, Samuel W. Lambert
Medical Jurisprudence, Israel Strauss
III. Review of Academy activities of the past eight years and plans for the future, Linsly R. Williams, Director

Thursday Evening, January 21, at 8:30 o'clock

THE FOURTH HARVEY LECTURE

"INVESTIGATIONS ON THE UNDERLYING CAUSES OF DEAFNESS"

S. J. CROWE
Baltimore

This lecture takes the place of the second Stated Meeting of the Academy for January.

SECTION MEETINGS

SECTION OF DERMATOLOGY AND SYPHILOLOGY
Tuesday Evening, January 5, at 8:30 o'clock

ORDER

I. READING OF THE MINUTES
II. PRESENTATION OF CASES
a. Cases from the New York Post-Graduate Medical School and Hospital
b. Miscellaneous cases
III. GENERAL DISCUSSION
IV. EXECUTIVE SESSION

SECTION OF NEUROLOGY AND PSYCHIATRY
Joint Meeting with the
NEW YORK NEUROLOGICAL SOCIETY
Tuesday Evening, January 5, at 8:30 o'clock

ORDER

I. READING OF THE MINUTES
II. PAPER OF THE EVENING
Basophilic adenomas and their clinical syndrome, Harvey Cushing, Professor of Surgery, Harvard Medical School

SECTION OF SURGERY
Friday Evening, January 8, at 8:30 o'clock

ORDER

I. READING OF THE MINUTES
II. PRESENTATION OF CASES
a. 1. A case of giant celled tumor of left upper jaw. Excision of tumor
2. A case of carcinoma of right upper jaw. Resection of maxilla
3. A case of sarcoma of floor of the mouth involving left lower jaw
a. Resection of tumor with a portion of mandible
b. Restoration of continuity of mandible with rib graft, William F. MacFee
b. Two cases of compound fracture, both bones of legs; immediate debridement, suture and cast, Philip C. Potter
c. 1. A case of esophageal diverticulum; two-stage operation
2. A case of recurrent pneumococcus peritonitis
3. A case of congenital tracheo-esophageal fistula; x-rays and specimens, John V. Bohrer
d. Diabetic arteriosclerotic gangrene of foot:
1. Modified guillotine amputation lower third of leg. Patient aged 70
2. True guillotine amputation lower third right leg; revision of stump one year later. Modified guillotine amputation lower third left leg. Patient aged 45
3. Circular flap amputation lower third right leg. Patient aged 70, Beverly C. Smith
III. PAPER OF THE EVENING
Modified guillotine amputation through lower third of leg for diabetic arteriosclerotic gangrene, Beverly C. Smith
IV. GENERAL DISCUSSION
V. EXECUTIVE SESSION

SECTION OF HISTORICAL AND CULTURAL MEDICINE
Wednesday Evening, January 13, at 8:30 o'clock

ORDER

I. READING OF THE MINUTES
II. PAPERS OF THE EVENING
a. An early undertaking in hospital social service, Marshall C. Pease, Jr.

b. The dark ages of psychiatric history, Gregory Zilboorg (by invitation)

III. General Discussion

IV. Executive Session

Section of Pediatrics

Thursday Evening, January 14, at 8:30 o'clock

ORDER

Symposium by Members of the Department of Pediatrics, Johns Hopkins Medical School

I. Effect of copper on iron metabolism, Hugh Josephs (by invitation)
Discussion, Randolph West

II. Prognosis of nephritis and nephrosis in children, Harriet Guild (by invitation), Vivian Tappan (by invitation)
Discussion, John D. Lyttle

III. Some aspects of mineral metabolism in parathyroidectomized animals, David Shelling (by invitation)
Discussion, Edwards A. Park (by invitation)

IV. Rocky mountain spotted fever in children, Paul G. Shipley (by invitation)
Discussion, Thomas M. Rivers

V. Studies of fat metabolism in infants, H. C. Tidwell (by invitation), C. M. Kirk (by invitation), L. E. Holt, Jr. (by invitation)
Discussion, Warren Sperry (by invitation)

Section of Orthopedic Surgery

Friday Evening, January 15, at 8:30 o'clock

ORDER

I. Reading of the Minutes

II. Paper of the Evening
Brain injuries at birth: their classification and subsequent treatment. Moving pictures, Winthrop M. Phelps, New Haven (by invitation)

III. General Discussion

IV. Executive Session

Section of Ophthalmology

Monday Evening, January 18, at 8:00 o'clock

From the Services of the N. Y. Post-Graduate & Knapp Memorial Hospitals

ORDER

I. Reading of the Minutes

II. Executive Session

III. Report of Cases

1. a. Hole in the macula
   b. True follicles in ciliary body
   c. Posterior ciliary veins
   Lantern slides, Louise Meeker (by invitation). Time 10 minutes
2. Lantern slide demonstration of muscle cases, Rudolf Aebli (by invitation). Time 10 minutes
   Discussion, J. W. White

3. a. Demonstration of a method of studying factors influencing the swelling of the vitreous
   b. Comparative studies of the chemical composition of the blood and cerebrospinal fluid in primary glaucoma, Martin Cohen, L. Halpern (by invitation), John A. Killian (by invitation). Time 10 minutes

IV. Paper of the Evening

Relation of human constitution to disease, George Draper

Report of Cases

4. Demonstration of brain with a pituitary tumor, John A. Billingsley (by invitation). Time 5 minutes
5. Treatment of toxic amblyopia with vasodilators, W. F. Duggan (by invitation). Time 5 minutes
6. Cases of retinal detachment treated by the Gonin method, Franklin Bracken (by invitation)
7. Radium injury to the conjunctiva, Arnold Knapp. Time 10 minutes
8. Demonstration of a method of registering ocular pulsations, Benjamin Friedman (by invitation). Time 5 minutes
9. a. A case of angioid streaks of retina (demonstration at the Gullstrand ophthalmoscope)
   b. A case of granuloma of cornea with unusual features, David A. Newman. Time 5 minutes
10. a. A case of Paget's Disease
    b. A case of glaucoma associated with naevus of face and globe (slit lamp demonstration), H. H. Tyson. Time 10 minutes
11. Slit lamp demonstration of a case of lentiglobus posterior, Joseph Ziporkes (by invitation)
12. A case of ankylo blepharon (familial, bilateral) with operative results, A. Fine (by invitation)
13. Radium therapy in the N. Y. Eye & Ear Infirmary, G. A. Robinson. Time 10 minutes

To facilitate the examination of the patients the demonstration began at 8 P. M.

Section of Medicine

Tuesday Evening, January 19, at 8:30 o'clock

ORDER

Papers of the Evening

I. Bronchial asthma, Maximilian A. Ramirez
II. Infective asthma, Robert A. Cooke
III. Infective asthma, R. Clark Grove (by invitation)
IV. General Discussion

Section of Genito-Urinary Surgery

Wednesday Evening, January 20, at 8:30 o'clock

ORDER

I. Reading of the Minutes

II. Papers of the Evening
   a. Endocrine factors involved in the development of the genital system
      Lantern slide illustrations, William Engelbach (by invitation)
   b. Impotence, its diagnosis and treatment, Max Huhner
   Discussion opened by Irving Pardee, James I. Farrell (by invitation)
III. General Discussion

Section of Oto-Laryngology
Wednesday Evening, January 20, at 8:30 o'clock

ORDER

I. Reading of the Minutes
II. Presentation of Cases
III. Papers of the Evening
   Evaluation of roentgenology in oto-laryngology
   a. Anatomy of the sinuses and mastoids, Mr. E. Burchell (by invitation)
   b. Teeth, Solomon Fineman
   c. Sinuses, Frederick M. Law
   d. Mastoid, George S. Dixon
   e. Larynx, Ross Golden
   f. Bronchi, Leon T. LeWald
   g. Oesophagus, A. Judson Quimby
   h. Chest, Leopold Jaches
IV. Discussion
   Harmon Smith, Duncan Macpherson, Mervin C. Myerson, Bissell B. Palmer, Luther B. MacKenzie
V. Executive Session

Section of Obstetrics and Gynecology
Tuesday Evening, January 26, at 8:30 o'clock

ORDER

I. Reading of the Minutes
II. Presentation of Cases
   Program presented by the Staff of Mt. Sinai Hospital
   a. Post menopausal bleeding, Samuel H. Geist, discussion, Gerard L. Moench
   b. The question of myomectomy in relation to fertility with some observations on technic, Isidor C. Rubin, discussion, Hiram N. Vineberg, Samuel J. Scadron
   c. Induction of therapeutic abortion by means of the Roentgen rays, William Harris (by invitation), discussion, Hiram N. Vineberg
   d. Ventral hernia following ventrofixation, Howard E. Lindeman, Max D. Mayer (by invitation)
   e. Ureter stricture as a factor in the diagnosis of pelvic disturbances —2 cases, Isidor Kross, discussion, Gerard L. Moench

f. Kraurosis vulvae and its treatment
Report of 12 cases, Morris A. Goldberg, discussion, Theodore Neustaedter (by invitation)
g. Treatment of dyspareunia, Max D. Mayer (by invitation), Hiram N. Vineberg

III. General Discussion
IV. Executive Session

AFFILIATED SOCIETIES

New York Meeting of the Society for Experimental Biology and Medicine Under the auspices of The New York Academy of Medicine

Wednesday Evening, January 20, at 8:15 o'clock

I. A new method for staining bacterial capsules, J. W. Churchman and N. V. Emelianoff
II. Presence of capsules on "non-capsulated" microorganisms, J. W. Churchman and N. V. Emelianoff
III. Studies of yellow fever virus in tissue culture, E. Haagen and M. Theiler (introduced by J. H. Bauer)
IV. Lethal action in the short-tailed mutation in the house mouse, Paul Chesley (introduced by L. C. Dunn)
V. Specific and non-specific cell polysaccharides of the human type tubercle bacillus, M. Heidelberger and A. E. O. Menzel
VI. Protein fractions of the timothy grass bacillus, M. Heidelberger and A. E. O. Menzel
VII. Influence of ortho-substitution on the conjugation of benzoic acid, A. J. Quick
VIII. General hyperthermia with heat localization by radiothermy, W. Bierman and M. Schwarzschild (introduced by B. N. Berg)

New York Pathological Society In Affiliation with The New York Academy of Medicine

Thursday Evening, January 28, at 8:30 o'clock

ORDER

I. Papers of the Evening
a. Red cell regeneration following copperin therapy, Burr R. Whitcher
b. Renal blastoma with cranial metastasis, J. S. Grewal (by invitation)
c. The tonsil in relation to branchio-genetic cysts, Louise Meeker
d. The influence of blood and of exudate upon the action of bacteriophage against the colon bacillus, Martha Applebaum (by invitation), Ward J. MacNeal

e. Studies on commercial bacteriophage products, Martha Straub (by invitation), Martha Applebaum (by invitation)

f. Bacteriophage as a therapeutic agent in staphylococcus bacteremia, Ward J. MacNeal, Frances C. Frisbee (by invitation)

II. Executive Session
Election of Officers

The New York Roentgen Society
in affiliation with
The New York Academy of Medicine
The New York Roentgen Society
invited
The New England Roentgen Ray Society,
The Philadelphia Roentgen Ray Society
and
The Roentgenologists of Baltimore and Washington
to attend a
Roentgenological Conference
Friday, January 29
and
Saturday, January 30
Friday morning conference at The Presbyterian Hospital
Friday afternoon conference at The Mt. Sinai Hospital
Saturday morning conference at The Memorial Hospital
Saturday afternoon conference at The New York Academy of Medicine

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FELLOWS ELECTED FEBRUARY 4, 1932

Simon Anthony Beisler.....121 East 60 Street
John Woolman Churchman .....215 East 72 Street
John Dorsey Craig .....12 East 11 Street
Gilman Sterling Currier .....Bernardsville, N. J.
John Staige Davis.....791 Park Avenue
William Robert Delzell.....945 Lexington Avenue
Alan Gregg ..... 61 Broadway
Edward Bellamy Gresser.....39 Fifth Avenue
Sophia J. Kleegman ..... 59 East 54 Street
William Andrew Krieger .....Poughkeepsie, N. Y.
Henry Minsky .....1 West 86 Street
Samuel Torrey Orton .....66 East 79 Street
Louis Apgar Pyle ..... Jersey City
Edward J. Riley .....102 East 68 Street
Irving R. Roth .....45 East 82 Street
Seth Selig .....18 West 82 Street
Lawrence Wells Sloan .....620 West 168 Street
Herbert Edward Stein .....41 West 75 Street

Carl TenBroeck ..... Rockefeller Institute, Princeton
Norman Treves ..... 155 East 72 Street
George Barnet Trible .....Washington, D. C.
Arthur Fulton Warren .....50 East 63 Street
Irving Sherwood Wright ..... 525 Park Avenue
Frederick A. Wurzbach, Jr. .....2600 Creston Avenue
Asher Yaguda ..... Newark

AND FOR ASSOCIATE FELLOWSHIP

Walther Friedrich Goebel, Ph.D. .....Rockefeller Institute

DEATHS OF FELLOWS OF THE ACADEMY

George Henry Fox, M.D., 1 Mather Street, Binghamton, N. Y.; graduated in medicine from the University of Michigan, Ann Arbor, in 1909; elected a Fellow of The New York Academy of Medicine April 4, 1929; died, December 26, 1931. Dr. Fox was a Fellow of the American Medical Association, a member of the County and State Medical Societies, a member of the American Society for Clinical Pathology, a member of the Academy of Medicine of Binghamton, Dermatologist to City Hospital and Consulting Dermatologist to City Hospital, O.P.D.

Henry Illoway, M.D., 1113 Madison Avenue, New York City; graduated in medicine from the University of Cincinnati, in 1869; elected a Fellow of the Academy March 15, 1900; died, January 15, 1932. Dr. Illoway was a Fellow of the American Medical Association, a member of the County and State Medical Societies and other professional organizations.

Louis Livingston Seaman, M.D., Bronxville, N. Y.; graduated in medicine from Jefferson College, Philadelphia, Pa., in 1876; elected a Fellow of the Academy April 2, 1885; died, January 31, 1932. Dr. Seaman was a Fellow of the American Medical Association, a member of the County and State Medical Societies and many other professional organizations. In 1909 Dr. Seaman delivered the Anniversary Discourse of the Academy, on the subject "Personal Observations of the Sleeping Sickness in Uganda, with illustrations of a Short Safari to the Source of the Nile."